In the specification:

Please replace the sequence listing with the sequence listing attached hereto.

Please replace paragraph 84 on page 25 with the following:

-- **[0084]** Crystal structure of BoNT/A-Allergan shows the potential sites of N-glycosylation on the surface as follows: 173-NLTR (SEQ ID NO: 106), 382-NYTI (SEQ ID NO: 107), 411-NFTK (SEQ ID NO: 108), 417-NFTG (SEQ ID NO: 109), 971-NNSG (SEQ ID NO: 110), 1010-NISD (SEQ ID NO: 111), 1198-NASQ (SEQ ID NO: 112), 1221-NLSQ (SEQ ID NO: 113). In some embodiments, g-BoNT/A (including g-iBoNT/A) is glycosylated at 173-NLTR (SEQ ID NO: 106), 382-NYTI (SEQ ID NO: 107), 411-NFTK (SEQ ID NO: 108), 417-NFTG (SEQ ID NO: 109), 971-NNSG (SEQ ID NO: 110), 1010-NISD (SEQ ID NO: 111), 1198-NASQ (SEQ ID NO: 112) and/or 1221-NLSQ (SEQ ID NO: 113). Potential sites of N-glycosylation for BoNT/E are as follows: 97-NLSG (SEQ ID NO: 114), 138-NGSG (SEQ ID NO: 115), 161-NSSN (SEQ ID NO: 116), 164-NISL (SEQ ID NO: 117), 365-NDSI (SEQ ID NO: 118), and 370-NISE (SEQ ID NO: 119). In some embodiments, g-BoNT/E (including g-iBoNT/E) is glycosylated at 97-NLSG (SEQ ID NO: 114), 138-NGSG (SEQ ID NO: 115), 161-NSSN (SEQ ID NO: 116), 164-NISL (SEQ ID NO: 117), 365-NDSI (SEQ ID NO: 118), and/or 370-NISE (SEQ ID NO: 119).--

Please replace paragraph 87 on page 26 with the following:

-- **[0087]** In some embodiments, the g-BoNT/A or g-iBoNT/A is about 150 kDa, and the glycosylation adds about 20 to 30 kDa to the protein. In some embodiments, the g-BoNT/A or the g-iBoNT/A has about eight to twelve $Glc_3Man_9GlcNAc_2$ (molecular weight of about 2600 dalton). In some embodiments, the g-BoNT/A or g-iBoNT/A is glycosylated with $Glc_3Man_9GlcNAc_2$ at positions 173-NLTR (SEQ ID NO: 106), 382-NYTI (SEQ ID NO: 107), 411-NFTK (SEQ ID NO: 108), 417-NFTG (SEQ ID NO:

109), 971-NNSG (SEQ ID NO: 110), 1010-NISD (SEQ ID NO: 111), 1198-NASQ (SEQ ID NO: 112), 1221-NLSQ (SEQ ID NO: 113).

Please replace paragraph 127, beginning on page 37 and ending on page 38 with the following:

-- **[00127]** Full-length iBoNT/A, LC, iLC were subcloned into pBAC-1 or pBACgus-1 vectors. For construction of inactive LC and inactive BoNT/A, the point mutant H227Y at LC of BoNT/A has been shown to abolish LC activity. Therefore, to make inactive full-length BoNT/A, we have introduced the mutant H227Y by PCR with the site mutagenesis QuickChange XL kit (Stratagene, Calif.). The mutagenic oligonucleotide primers have been designed individually according to the desired mutation. The sense primer is 5'-GTA ACA TTA GCA CAT GAA CTT ATA TAT GCT GGA CAT AGA TTA TAT GGA ATA GCA ATT-3' (SEQ ID NO: 120). The antisense primer is 5'-AAT TGC TAT TCC ATA TAA TCT ATG TCC AGC ATA TAT AAG TTC ATG TGC TAA TGT TAC-3' (SEQ ID NO: 120). The positive clones were selected and confirmed by restriction enzymes digestion and DNA sequencing.